STIC Search Report
EIC 1700

STIC Database Tracking Number: 157900

TO:	From:
Ben Sackey **Location: REM 5B31** **Art Unit : 1626** **July 19, 2005** **Case Serial Number: 10/618578**	**Kathleen Fuller** **Location: EIC 1700** **REMSEN 4B28** **Phone: 571/272-2505** **Kathleen.Fuller@uspto.gov**

Search Notes

This does not seem to be structurally searchable. The compounds indexed to the applicant are all manually indexed have no structures associated with the registry numbers I used the RN's and a text search for the attached.

Access DB# 157900

SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: BEN SACKET Examiner #: 73489 Date: 6/28/05
Art Unit: 1626 Phone Number ~~30~~ 2-0704 Serial Number: 10/618,578
Mail Box and Bldg/Room Location: REM 5 331 Results Format Preferred (circle): PAPER DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need.

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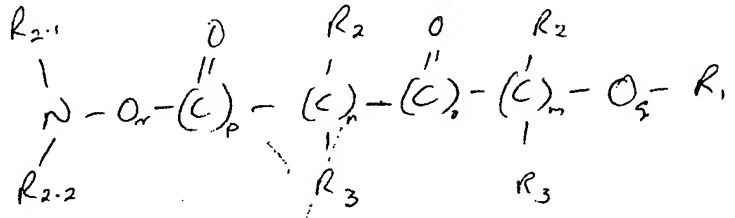
Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: Compounds for the modulation of the glycolysis enzyme transaminase Complex and/or

Inventors (please provide full names): Eigenbrodt et al.

Earliest Priority Filing Date: 09/11/02

For Sequence Searches Only *Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*



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STAFF USE ONLY	Type of Search		Vendors and cost where applicable	
Searcher: X. Fuller	NA Sequence (#)		STN ✓	
Searcher Phone #:	AA Sequence (#)		Dialog	
Searcher Location:	Structure (#)	10	Questel/Orbit	
Date Searcher Picked Up:	Bibliographic		Dr.Link	
Date Completed: 7/19/05	Litigation		Lexis/Nexis	
Searcher Prep & Review Time: 40	Fulltext		Sequence Systems	
Clerical Prep Time:	Patent Family		WWW/Internet	
Online Time: 32	Other		Other (specify)	

PTO-1590 (8-01)

=> FILE HCAPLUS
FILE 'HCAPLUS' ENTERED AT 16:37:55 ON 19 JUL 2005

FILE COVERS 1907 - 19 Jul 2005 VOL 143 ISS 4
FILE LAST UPDATED: 18 Jul 2005 (20050718/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> D ALL L49

L49 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:218483 HCAPLUS
DN 140:252402
ED Entered STN: 19 Mar 2004
TI Glycolysis and/or transaminase complex modulators for the treatment of different diseases
IN Eigenbrodt, Erich; Scheefers, Hans; Mazurek, Sybille
PA ScheBo Biotech A.-G., Germany
SO Ger. Offen., 8 pp.
CODEN: GWXXBX
DT Patent
LA German
IC ICM C07C239-22
ICS C07C261-02; C07C327-00
CC 16-2 (Fermentation and Bioindustrial Chemistry)
Section cross-reference(s): 1, 23

applicant

FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 10244080	A1	20040318	DE 2002-10244080	20020906
CA 2498045	AA	20040325	CA 2003-2498045	20030707
WO 2004024676	A1	20040325	WO 2003-DE2344	20030707
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1534666	A1	20050601	EP 2003-794769	20030707

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

US 2004147587 A1 20040729 US 2003-618578 20030711 <--

PRAI DE 2002-10244080 A 20020906
DE 2002-10242445 A 20020911
DE 2002-10244299 A 20020923
WO 2003-DE2344 W 20030707

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
DE 10244080	ICM	C07C239-22
	ICS	C07C261-02; C07C327-00
DE 10244080	ECLA	C07D261/18
WO 2004024676	ECLA	C07D261/18
US 2004147587	NCL	514/417.000; 514/528.000; 514/506.000; 548/479.000; 558/232.000
	ECLA	C07D261/18 <--

OS MARPAT 140:252402

AB The invention concerns compds. for the modulation glycolysis of enzyme complex and the transaminase complex, pharmaceutical compns. containing such compds. as well as uses from such compds. to the production from pharmaceutical compns. to the treatment of different diseases.

ST transaminase glycolysis enzyme modulator

IT Inflammation
(Crohn's disease, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Intestine, disease
(Crohn's, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Kidney, disease
(Goodpasture's syndrome, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Animal cell line
(MCF-7; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Animal cell line
(Novikoff-Hepatic; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Disease, animal
(adipose tissue, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Autoimmune disease
(autoimmune thrombocytopenia, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Autoimmune disease
Inflammation
Thyroid gland, disease
(autoimmune thyroiditis, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Infection
(bacterial, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Human
(cells and enzymes; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Arthritis
(chronic, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)

IT Cartilage, disease
(degeneration, use to treat; glycolysis and/or transaminase complex

modulators for treatment of different diseases)
IT Platelet (blood)
(disease, autoimmune thrombocytopenia, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Joint, anatomical
(disease, degeneration, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Adipose tissue
(disease, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Drugs
(glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Intestine, disease
(inflammatory, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Cell proliferation
(inhibition; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Drug delivery systems
(injections, i.v.; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Autoimmune disease
(insulin-dependent diabetes mellitus, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Diabetes mellitus
(insulin-dependent, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Disease, animal
(joint degeneration, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Glycolysis
(modulation of; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Drug delivery systems
(oral; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Arthritis
(polyarthritis, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Asthma
Autoimmune disease
Cachexia
Connective tissue, disease
Diabetes insipidus
Multiple sclerosis
Myasthenia gravis
Psoriasis
Sepsis
(use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT Eye, disease
Inflammation
(uveitis, use to treat; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT 9031-66-7, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(complex; glycolysis and/or transaminase complex modulators for treatment of different diseases)
IT 9000-97-9, Glutamate oxaloacetate transaminase 9001-47-2, Glutaminase

9001-59-6, Pyruvate kinase 9001-64-3, Malate dehydrogenase 9014-27-1, Serine dehydratase 9015-68-3, Asparaginase 9026-51-1, Nucleotide diphosphate kinase 9032-62-6, Phosphoglyceromutase 9067-84-9, Deaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (glycolysis and/or transaminase complex modulators for treatment of different diseases)

=> FILE REG
FILE 'REGISTRY' ENTERED AT 16:38:36 ON 19 JUL 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 18 JUL 2005 HIGHEST RN 855828-45-4
DICTIONARY FILE UPDATES: 18 JUL 2005 HIGHEST RN 855828-45-4

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 18, 2005

Please note that search-term pricing does apply when conducting SmartSELECT searches.

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* *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added, *
* effective March 20, 2005. A new display format, IDERL, is now *
* available and contains the CA role and document type information. *
* *
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Structure search iteration limits have been increased. See HELP SLIMITS for details.

Experimental and calculated property data are now available. For more information enter HELP PROP at an arrow prompt in the file or refer to the file summary sheet on the web at:
http://www.cas.org/ONLINE/DBSS/registryss.html

=> D HIS L50

(FILE 'HCAPLUS, METADEX' ENTERED AT 15:56:33 ON 19 JUL 2005)
SEL RN

FILE 'REGISTRY' ENTERED AT 16:15:53 ON 19 JUL 2005
L50 10 S E1-E10

Registry numbers from the applicant no structures

=> D L50 1-10

L50 ANSWER 1 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9067-84-9** REGISTRY
ED Entered STN: 16 Nov 1984
CN Deaminase (9CI) (CA INDEX NAME)
MF Unspecified

CI MAN
LC STN Files: AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CEN, CHEMLIST, CIN, EMBASE, IFICDB, IFIPAT, IFIUDB, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
253 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
253 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 2 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9032-62-6** REGISTRY
ED Entered STN: 16 Nov 1984
CN Phosphomutase, glycerate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Bisphosphoglyceromutase
CN Cofactor-independent phosphoglycerate mutase
CN Diphosphoglycerate mutase
CN Diphosphoglyceric mutase
CN Diphosphoglycomutase
CN E.C. 2.7.5.3
CN E.C. 5.4.2.1
CN Glycerate phosphomutase
CN Glycerate phosphomutase (diphosphoglycerate cofactor)
CN Monophosphoglycerate mutase
CN Monophosphoglyceromutase
CN Phosphoglycerate mutase
CN Phosphoglycerate phosphomutase
CN Phosphoglyceric acid mutase
CN Phosphoglyceromutase
DR 9023-91-0
MF Unspecified
CI MAN
LC STN Files: AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMLIST, CSCHEM, EMBASE, MSDS-OHS, NAPRALERT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1155 REFERENCES IN FILE CA (1907 TO DATE)
18 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1158 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 3 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9031-66-7** REGISTRY
ED Entered STN: 16 Nov 1984
CN Aminotransferase (9CI) (CA INDEX NAME)
OTHER NAMES:
CN α-Aminotransferase
CN α-Oxoglutaric acid transaminase
CN Glutamate aminotransferase
CN L-Amino acid aminotransferase
CN Transaminase
DR 9012-55-9

MF Unspecified
CI COM, MAN
LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CEN, CIN, CSNB, EMBASE, IFICDB, IFIPAT, IFIUDB, NAPRALERT, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
2444 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2445 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 4 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9026-51-1** REGISTRY
ED Entered STN: 16 Nov 1984
CN Kinase (phosphorylating), nucleoside diphosphate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN CDP kinase
CN Diphosphonucleoside kinase
CN E.C. 2.7.4.6
CN Nucleoside 5'-diphosphate kinase
CN Nucleoside diphosphate (UDP) kinase
CN Nucleoside diphosphate kinase
CN Nucleoside diphosphokinase
CN Nucleotide diphosphate kinase
CN UDP kinase
CN Uridine diphosphate kinase
MF Unspecified
CI MAN
LC STN Files: AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CSCHEM, EMBASE, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1274 REFERENCES IN FILE CA (1907 TO DATE)
25 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1279 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 5 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9015-68-3** REGISTRY
ED Entered STN: 16 Nov 1984
CN Asparaginase (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN α-Asparaginase
CN Colaspase
CN Crasnitin
CN Crisantaspase
CN E.C. 3.5.1.1
CN Elspar
CN Erwinase
CN Kidrolase
CN L-Asnase
CN L-Asparaginase
CN L-Asparagine amidohydrolase
CN Leunase
CN MK 965
CN NSC 109229

CN Oncospar
DR 9037-33-6, 9037-34-7, 9060-77-9
MF Unspecified
CI COM, MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MRCK*, NAPRALERT, NIOSHTIC, PHAR, PROMT, PS, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3134 REFERENCES IN FILE CA (1907 TO DATE)
200 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3140 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 6 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9014-27-1** REGISTRY
ED Entered STN: 16 Nov 1984
CN Dehydratase, L-serine (9CI) (CA INDEX NAME)
OTHER NAMES:
CN E.C. 4.2.1.13
CN L-Hydroxy amino acid dehydratase
CN L-Serine deaminase
CN L-Serine dehydratase
CN Serine deaminase
CN Serine dehydratase
DR 9014-28-2
MF Unspecified
CI MAN
LC STN Files: AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CIN, EMBASE, TOXCENTER, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

670 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
670 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 7 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9001-64-3** REGISTRY
ED Entered STN: 16 Nov 1984
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 37,000-Mol.-wt. adrenergically induced proteins, pI 6.0
CN Adrenergically induced proteins, 37,000-mol.-wt., pI 6.0
CN AIP 37/6 proteins
CN E.C. 1.1.1.37
CN L-Malate dehydrogenase
CN Malate (NAD) dehydrogenase
CN Malate dehydrogenase
CN Malate dehydrogenase (NAD)
CN Malate dehydrogenase NAD-dependent

CN Malic acid dehydrogenase
CN Malic dehydrogenase
CN MDH
CN NAD-dependent malate dehydrogenase
CN NAD-dependent malic dehydrogenase
CN NAD-L-malate dehydrogenase
CN NAD-linked malate dehydrogenase
CN NAD-malate dehydrogenase
CN NAD-malic dehydrogenase
CN NAD-specific malate dehydrogenase
CN PI 6.0 adrenergically induced proteins, 37,000-mol.-wt.
CN Proteins, adrenergically induced, 37,000-mol.-wt., pI 6.0
CN Proteins, AIP 37/6
CN Proteins, AIP 37/6 (adrenergically induced protein, 37,000-mol.-wt., pI 6.0)
MF Unspecified
CI MAN
LC STN Files: AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MSDS-OHS, NIOSHTIC, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

11319 REFERENCES IN FILE CA (1907 TO DATE)
156 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
11327 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 8 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9001-59-6** REGISTRY
ED Entered STN: 16 Nov 1984
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN E.C. 2.7.1.40
CN Fluorokinase
CN Kinase (phosphorylating), fluoro-
CN Phosphoenolpyruvate kinase
CN Pyruvate kinase
CN pyruvate phosphotransferase (EC 2.7.1.40)
CN Pyruvic kinase
MF Unspecified
CI MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MSDS-OHS, NIOSHTIC, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

7341 REFERENCES IN FILE CA (1907 TO DATE)
83 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
7350 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 9 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9001-47-2** REGISTRY
ED Entered STN: 16 Nov 1984
CN Glutaminase (9CI) (CA INDEX NAME)
OTHER NAMES:
CN E.C. 3.5.1.2
CN Glutaminase C 100S
CN Glutaminase C 200
CN Glutaminase Daiwa C-100
CN Glutaminase Daiwa C100S
CN Glutaminase FP
CN Glutaminase I
CN Glutamine aminohydrolase
CN L-Glutaminase
CN L-Glutamine deaminase
CN Y 600S
MF Unspecified
CI COM, MAN
LC STN Files: AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, MEDLINE, NAPRALERT, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1964 REFERENCES IN FILE CA (1907 TO DATE)
24 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1966 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L50 ANSWER 10 OF 10 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9000-97-9** REGISTRY
ED Entered STN: 16 Nov 1984
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2-Oxoglutarate-glutamate aminotransferase
CN Aspartate α-ketoglutarate transaminase
CN Aspartate aminotransferase
CN Aspartate-2-oxoglutarate transaminase
CN Aspartic acid aminotransferase
CN Aspartic aminotransferase
CN Aspartyl aminotransferase
CN AST
CN E.C. 2.6.1.1
CN Glutamate 2-oxoglutarate transaminase
CN Glutamate-oxalacetate aminotransferase
CN Glutamate-oxalate transaminase
CN Glutamate-oxaloacetate transaminase
CN Glutamic-aspartic aminotransferase
CN Glutamic-aspartic transaminase
CN Glutamic-oxalacetic transaminase
CN Glutamic-oxalic transaminase
CN GOT
CN GOT (enzyme)
CN L-Aspartate aminotransferase
CN L-Aspartate transaminase
CN L-Aspartate-α-ketoglutarate transaminase

CN L-Aspartate-2-ketoglutarate aminotransferase
CN L-Aspartate-2-oxoglutarate aminotransferase
CN L-Aspartate-2-oxoglutarate-transaminase
CN L-Aspartic aminotransferase
CN Oxalacetate-aspartate aminotransferase
CN Oxaloacetate transferase
CN SGOT
DR 9013-64-3, 9014-29-3, 9016-19-7, 9036-26-4, 9061-83-0, 61461-53-8, 139074-52-5
MF Unspecified
CI MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MSDS-OHS, NAPRALERT, NIOSHTIC, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

21950 REFERENCES IN FILE CA (1907 TO DATE)
125 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
21982 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> => FILE HCAPLUS
FILE 'HCAPLUS' ENTERED AT 16:51:57 ON 19 JUL 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 19 Jul 2005 VOL 143 ISS 4
FILE LAST UPDATED: 18 Jul 2005 (20050718/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> D QUE
L50 10 SEA FILE=REGISTRY ABB=ON (9000-97-9/BI OR 9001-47-2/BI OR 9001-59-6/BI OR 9001-64-3/BI OR 9014-27-1/BI OR 9015-68-3/BI OR 9026-51-1/BI OR 9031-66-7/BI OR 9032-62-6/BI OR 9067-84-9/BI)
L54 133 SEA FILE=HCAPLUS ABB=ON MODULAT?(6A)(GLYCOLYSIS? OR TRANSAMINASE?)
L55 23 SEA FILE=HCAPLUS ABB=ON L54 AND PHARMAC?/SC,SX

KATHLEEN FULLER EIC 1700 REMSON 4B28 571/272-2505

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L56          13 SEA FILE=HCAPLUS ABB=ON  L54 AND THU/RL
L57          25 SEA FILE=HCAPLUS ABB=ON  L55 OR L56
L59       47026 SEA FILE=HCAPLUS ABB=ON  L50
L61          27 SEA FILE=HCAPLUS ABB=ON  L54 AND L59
L62          11 SEA FILE=HCAPLUS ABB=ON  L61 AND (THU/RL OR PHARMAC?/SC,SX)
L63          25 SEA FILE=HCAPLUS ABB=ON  L57 OR L62


=> D L63 BIB ABS IND HHITSTR 1-25
'HHITSTR' IS NOT A VALID FORMAT FOR FILE 'HCAPLUS'

The following are valid formats:

ABS --------- GI and AB
ALL --------- BIB, AB, IND, RE
APPS -------- AI, PRAI
BIB --------- AN, plus Bibliographic Data and PI table (default)
CAN --------- List of CA abstract numbers without answer numbers
CBIB -------- AN, plus Compressed Bibliographic Data
DALL -------- ALL, delimited (end of each field identified)
DMAX -------- MAX, delimited for post-processing
FAM --------- AN, PI and PRAI in table, plus Patent Family data
FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, IPC, and NCL

IABS -------- ABS, indented with text labels
IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IMAX -------- MAX, indented with text labels
ISTD -------- STD, indented with text labels

OBIB -------- AN, plus Bibliographic Data (original)
OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs
```

OCC --------- Number of occurrence of hit term and field in which it occurs

To display a particular field or fields, enter the display field codes. For a list of the display field codes, enter HELP DFIELDS at an arrow prompt (=>). Examples of formats include: TI; TI,AU; BIB,ST; TI,IND; TI,SO. You may specify the format fields in any order and the information will be displayed in the same order as the format specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR, FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):END

=> D BIB ABS IND HITSTR 1-25

L63 ANSWER 1 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2005:523402 HCAPLUS
DN 143:60254
TI Preparation of compounds for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease
IN Scheefers, Hans
PA ScheBo-Biotech AG, Germany
SO PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005054174	A2	20050616	WO 2004-DE2691	20041206
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10357301	A1	20050707	DE 2003-10357301	20031205
PRAI	DE 2003-10357301	A	20031205		

AB The invention relates to compds. for **modulating** the **glycolysis** enzyme complex and the **transaminase** complex, pharmaceutical compns. containing said compds., and to the uses of said compds. for the production of pharmaceutical compns. for the treatment of different illnesses. A discussion example gives the preparation of 5-oxyamino-2-aminopentanoic acid from 5-hydroxy-2-aminopentanoic acid (no data). Four figures present compds. typifying the claimed compds. (no data).
IC ICM C07C229-00
CC 34-3 (Amino Acids, Peptides, and Proteins)
Section cross-reference(s): 1, 33
ST **glycolysis** enzyme **modulator** prepn treatment disease
IT Inflammation
(Crohn's disease; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Intestine, disease
(Crohn's; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Kidney, disease
(Goodpasture's syndrome; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Autoimmune disease
(autoimmune thrombocytopenia; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Autoimmune disease
Inflammation
Thyroid gland, disease
(autoimmune thyroiditis; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Infection
(bacterial; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Infection
(chronic; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(deaminases; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Platelet (blood)
(disease, autoimmune thrombocytopenia; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Joint, anatomical
(disease, degeneration; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Heart, disease
(failure, chronic; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Heart, disease
(failure; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Intestine, disease
(inflammatory; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Autoimmune disease
(insulin-dependent diabetes mellitus; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Diabetes mellitus
(insulin-dependent; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)
IT Disease, animal

(joint degeneration; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Peptides, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligopeptides; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Arthritis
(polyarthritis, chronic; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Acidosis
Arthritis
Asthma
Autoimmune disease
Cachexia
Connective tissue, disease
Diabetes insipidus
Glycolysis
Multiple sclerosis
Myasthenia gravis
Neoplasm
Osteoarthritis
Psoriasis
Rheumatic diseases
Sepsis
(preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Amino acids, reactions
Hydroxylamines
RL: RCT (Reactant); **THU (Therapeutic use)**; BIOL (Biological study); RACT (Reactant or reagent); USES (Uses)
(preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Nucleosides, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease from)

IT Eye, disease
Inflammation
(uveitis; preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of disease)

IT 9000-86-6, Glutamate pyruvate **transaminase 9000-97-9**
9001-46-1, Glutamate dehydrogenase **9001-59-6**, Pyruvate kinase
9001-64-3, Malate dehydrogenase **9014-27-1**
9015-68-3, Asparaginase **9031-66-7**, **Transaminase**
9032-62-6, Phosphoglyceromutase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of compds. for **modulating** the **glycolysis** enzyme complex and/or the transaminase complex for treatment of

disease)
IT **9000-97-9 9001-59-6**, Pyruvate kinase **9001-64-3**
, Malate dehydrogenase **9014-27-1 9015-68-3**,
Asparaginase **9031-66-7**, **Transaminase 9032-62-6**
, Phosphoglyceromutase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of compds. for **modulating** the **glycolysis**
enzyme complex and/or the transaminase complex for treatment of
disease)
RN 9000-97-9 HCAPLUS
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9014-27-1 HCAPLUS
CN Dehydratase, L-serine (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9015-68-3 HCAPLUS
CN Asparaginase (8CI, 9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9032-62-6 HCAPLUS
CN Phosphomutase, glycerate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 2 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:681711 HCAPLUS
DN 141:167780
TI Reagents that modulate the activity of TLR9 and methods and compositions for the prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases
IN Liu, Ningshu; Watanabe, Shinichi; Ni, Lin; Bacon, Kevin
PA Bayer Healthcare A.-G., Germany
SO PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004070344	A2	20040819	WO 2004-EP641	20040127

W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX,

MZ, MZ, NA, NI
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI EP 2003-2183 A 20030204
EP 2003-21122 A 20030922

AB The effect of TLR-9 modulation can be detected by determining e.g., the amount of T-bet mRNA or protein, STAT4 protein phosphorylation, p38 activities, IL-12 mRNA or protein, inhibition of TH2-related IgG1 and IgE switching, present in a tissue. The present invention relates to method for identifying or evaluating reagents that modulate the activity of TLR9 using these members of the pathway such as T-bet, NF-B, IKK, STAT4, p38, IL-12 and Igs as markers. Reagents that modulate the activity of TLR9 identified by the present method are useful in the manufacture of medicaments for the treatment of a range of diseases including cancer, autoimmune diseases, inflammatory diseases such as asthma or COPD, immunol. disorders and any other conditions involving aberrations of signal transduction.

IC ICM G01N

CC 1-7 (**Pharmacology**)
Section cross-reference(s): 9

ST TLR9 prognosis diagnosis autoimmune disease inflammation asthma COPD

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (BATF, PLAUR, ARHE, TACSTD1, LMO4, FNBP3, GNPNAT1, B-cell BAP29, FIG1, CD74; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study) (C/EBP-β (CCAAT box/enhancer element-binding protein β); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study) (C/EBP-δ (CCAAT box/enhancer element-binding protein δ); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study) (EGR2, mouse; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Epidermal growth factor receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study) (ESP15; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study) (ID2 (inhibitor of differentiation 2); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Antibodies and Immunoglobulins
Immunoglobulin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(IgE; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IgG1; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IgG2a autoantibodies; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MyD88 (myeloid differentiation primary response protein 88); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PML; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT1 (signal transducer and activator of transcription 1); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT4 (signal transducer and activator of transcription 4); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT6 (signal transducer and activator of transcription 6); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TBX21 (T-box 21); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TCF (T-cell factor), TCF2; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(TLR-9 (Toll-like receptor-9); reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Lung, disease
(chronic obstructive; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT DNA
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with CpG; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Immunity
(disorder; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(nucleolar organizer-associated, NOP5/NOP58; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(p38; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Leukemia
(promyelocytic; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Anti-inflammatory agents
Antiasthmatics
Autoimmune disease
B cell (lymphocyte)
DNA microarray technology
Diagnosis
Inflammation
Signal transduction, biological
(reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Antibodies and Immunoglobulins
Interleukin 12
Interleukin 4
Polynucleotides
Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ubiquitin-activating, E1C; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment

of TLR9 related diseases)
IT 9014-36-2, GTP-specific succinyl-COA synthetase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(beta subunit; reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)
IT **9000-97-9**, Glutamate oxaloacetate **transaminase**
159606-08-3, IKK kinase 362516-16-3, Conserved helix-loop-helix ubiquitous kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reagents that **modulate** the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)
IT 2382-65-2D, conjugates with DNA
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(reagents that modulate the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)
IT **9000-97-9**, Glutamate oxaloacetate **transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reagents that **modulate** the activity of TLR9 and methods and compns. for prediction, diagnosis, prognosis, prevention and treatment of TLR9 related diseases)
RN 9000-97-9 HCAPLUS
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 3 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:620197 HCAPLUS
DN 141:179598
TI Antihyperlipidemic agent
IN Trifonova, O. Yu.; Khazanov, V. A.
PA Russia
SO Russ., No pp. given
CODEN: RUXXE7
DT Patent
LA Russian
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	RU 2228746	C1	20040520	RU 2002-131468	20021125
PRAI	RU 2002-131468		20021125		

AB The invention relates to antihyperlipidemic and antihyperproteinemic agents. The agent represents a combination of regulators of energetic metabolism, at least one represents succinic acid or its salt, either by combined using a known antihyperlipidemic agent in combination of some regulators of energetic metabolism or one taken in pharmacol. EDs. The invention provides an agent practically without adverse effects that can be used in treatment of atherosclerosis, ischemic heart disease and obesity.
IC ICM A61K031-194
ICS A61K031-191; A61K009-48; A61K009-22; A61P009-00
CC 63-6 (**Pharmaceuticals**)
ST hypolipemic antiatherosclerotic heart ischemia obesity
IT Antiarteriosclerotics
(antiatherosclerotics; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)
IT Antiobesity agents

Hypolipemic agents
Obesity
(antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT Drug delivery systems
(capsules; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT Ischemia
(cardiac; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT Heart, disease
(ischemia; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT Drug delivery systems
(tablets; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT 56-65-5, Atp, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT 56-86-0, Glutamic acid, biological studies 57-03-4, α Glycerophosphate 110-15-6, Succinic acid, biological studies 300-85-6, β Hydroxybutyric acid 320-77-4, Isocitric acid 328-50-7, α Ketoglutaric acid
RL: PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT 59-67-6, Nicotinic acid, biological studies 11041-12-6, Cholestyramine 23288-49-5, Probucol 50925-79-6, Colestipol
RL: PEP (Physical, engineering or chemical process); PYP (Physical process); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT 9028-35-7
RL: PEP (Physical, engineering or chemical process); PYP (Physical process); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(inhibitors, statins; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT 9002-02-2, Succinate dehydrogenase **9031-66-7, Transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**modulators**; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

IT **9031-66-7, Transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**modulators**; antihyperlipidemic agent for treatment of atherosclerosis, cardiac ischemia, and obesity)

RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 4 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:610042 HCAPLUS
DN 141:136207
TI Crystal structures of murine and human carnitine acyltransferases and

their uses in rational design of enzyme activity modulators
IN Tong, Liang; Jogl, Gerwald
PA The Trustees of Columbia University In the City of New York, USA
SO PCT Int. Appl., 390 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004062581	A2	20040729	WO 2004-US170	20040106

W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GH, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX, MZ

PRAI US 2003-438172P P 20030106

AB The present invention relates to structural models of carnitine acyltransferases, and, in particular, to models of the reactive sites of these enzymes. It is based, at least in part, on the x-ray crystallog. structures of murine carnitine acetyltransferase (mCRAT), both in pure form and in complex with its substrates carnitine and CoA. The structural information provides a basis for designing modulators of the activity of CRAT and related enzymes.

IC ICM A61K
CC 7-5 (Enzymes)
Section cross-reference(s): 1, 75
ST crystal structure carnitine acetyltransferase drug design; acyltransferase carnitine crystal structure drug design
IT Enzyme functional sites
(active; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT Conformation
Drug design
Drug screening
Human
Molecular modeling
Mus
(crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT Diabetes mellitus
(design of drugs for; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT Fatty acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(design of modulators of biosynthesis and oxidation of; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT **Glycolysis**
(design of **modulators** of; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT Antidiabetic agents
(design of; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)

IT Crystal structure
Molecular structure, natural product
(of murine and human carnitine acyltransferases)
IT 727435-76-9
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(amino acid sequence; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT 85-61-0D, Coenzyme A, complexes with carnitine acetyltransferase
541-15-1D, Carnitine, complexes with carnitine acetyltransferase
9029-90-7, Carnitine acetyltransferase 9029-90-7D, Carnitine acetyltransferase, complexes with carnitine or CoA 39386-49-7, Carnitine acyltransferase
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(design of modulators of sensitivity to; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)
IT 727439-76-1 727439-77-2
RL: PRP (Properties)
(unclaimed sequence; crystal structures of murine and human carnitine acyltransferases and their uses in rational design of enzyme activity modulators)

L63 ANSWER 5 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:253146 HCAPLUS
DN 140:269647
TI **Glycolysis** and/or **transaminase** complex **modulators** for the treatment of different diseases
IN Eigenbrodt, Erich; Scheefers, Hans; Mazurek, Sybille
PA Schebo Biotech Ag, Germany
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004024676	A1	20040325	WO 2003-DE2344	20030707
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10244080	A1	20040318	DE 2002-10244080	20020906
	CA 2498045	AA	20040325	CA 2003-2498045	20030707
	EP 1534666	A1	20050601	EP 2003-794769	20030707
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
PRAI	DE 2002-10244080	A	20020906		

DE 2002-10242445 A 20020911
DE 2002-10244299 A 20020923
WO 2003-DE2344 W 20030707

OS MARPAT 140:269647

AB The invention concerns compds. for the **modulation** **glycolysis** of enzyme complex and the **transaminase** complex, pharmaceutical compns. containing such compds. as well as uses from such compds. to the production from pharmaceutical compns. to the treatment of different diseases.

IC ICM C07C255-23
ICS C07C239-20; C07D261-18; A61K031-275; A61K031-21; A61P035-00

CC 16-2 (Fermentation and Bioindustrial Chemistry)
Section cross-reference(s): 1, 23

ST **transaminase** **glycolysis** enzyme **modulator**

IT Inflammation
(Crohn's disease, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Intestine, disease
(Crohn's, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Kidney, disease
(Goodpasture's syndrome, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Animal cell line
(MCF-7; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Animal cell line
(Novikoff-Hepatic; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Disease, animal
(adipose tissue, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Autoimmune disease
(autoimmune thrombocytopenia, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Autoimmune disease
Inflammation
Thyroid gland, disease
(autoimmune thyroiditis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Infection
(bacterial, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Human
(cells and enzymes; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Arthritis
(chronic, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Cytoplasm
(cytosol; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Cartilage, disease
(degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Platelet (blood)
(disease, autoimmune thrombocytopenia, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Joint, anatomical
(disease, degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Adipose tissue
(disease, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Antitumor agents
Drugs
Mitochondria
(**glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Intestine, disease
(inflammatory, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Cell proliferation
(inhibition; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Drug delivery systems
(injections, i.v.; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Autoimmune disease
(insulin-dependent diabetes mellitus, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Diabetes mellitus
(insulin-dependent, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Disease, animal
(joint degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT **Glycolysis**
(**modulation** of; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Drug delivery systems
(oral; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Arthritis
(polyarthritis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Asthma
Autoimmune disease
Cachexia
Connective tissue, disease
Diabetes insipidus
Multiple sclerosis

Myasthenia gravis
Psoriasis
Sepsis
(use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Eye, disease
Inflammation
(uveitis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT **9031-66-7**, Aminotransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(complex; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT 645-88-5 7803-49-8, Hydroxylamine, biological studies 9000-86-6, Glutamate pyruvate **transaminase 9000-97-9**, Glutamate oxaloacetate **transaminase 9001-47-2**, Glutaminase **9001-59-6**, Pyruvate kinase **9001-64-3**, Malate dehydrogenase **9014-27-1**, Serine dehydratase **9015-68-3**, Asparaginase **9026-51-1**, Nucleotide diphosphate kinase 9028-92-6, Glyceraldehyde-3-phosphate dehydrogenase 9029-12-3, Glutamate dehydrogenase 9029-83-8, Serine hydroxymethyltransferase 9030-88-0 9030-89-1, Serine **transaminase 9032-62-6**, Phosphoglyceromutase **9067-84-9**, Deaminase 75706-12-6 108605-62-5
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT **9031-66-7**, Aminotransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(complex; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **9000-97-9**, Glutamate oxaloacetate **transaminase** **9001-47-2**, Glutaminase **9001-59-6**, Pyruvate kinase **9001-64-3**, Malate dehydrogenase **9014-27-1**, Serine dehydratase **9015-68-3**, Asparaginase **9026-51-1**, Nucleotide diphosphate kinase **9032-62-6**, Phosphoglyceromutase **9067-84-9**, Deaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
RN 9000-97-9 HCAPLUS
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-47-2 HCAPLUS
CN Glutaminase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9014-27-1 HCAPLUS
CN Dehydratase, L-serine (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9015-68-3 HCAPLUS
CN Asparaginase (8CI, 9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9026-51-1 HCAPLUS
CN Kinase (phosphorylating), nucleoside diphosphate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9032-62-6 HCAPLUS
CN Phosphomutase, glycerate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9067-84-9 HCAPLUS
CN Deaminase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 6 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:218483 HCAPLUS
DN 140:252402
TI **Glycolysis** and/or **transaminase** complex **modulators** for the treatment of different diseases
IN Eigenbrodt, Erich; Scheefers, Hans; Mazurek, Sybille
PA ScheBo Biotech A.-G., Germany
SO Ger. Offen., 8 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10244080	A1	20040318	DE 2002-10244080	20020906
	CA 2498045	AA	20040325	CA 2003-2498045	20030707
	WO 2004024676	A1	20040325	WO 2003-DE2344	20030707
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1534666	A1	20050601	EP 2003-794769	20030707
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2004147587	A1	20040729	US 2003-618578	20030711
PRAI	DE 2002-10244080	A	20020906		
	DE 2002-10242445	A	20020911		
	DE 2002-10244299	A	20020923		
	WO 2003-DE2344	W	20030707		

OS MARPAT 140:252402

AB The invention concerns compds. for the **modulation** **glycolysis** of enzyme complex and the **transaminase** complex, pharmaceutical compns. containing such compds. as well as uses from such compds. to the production from pharmaceutical compns. to the treatment of different diseases.

IC ICM C07C239-22
ICS C07C261-02; C07C327-00

CC 16-2 (Fermentation and Bioindustrial Chemistry)
Section cross-reference(s): 1, 23

ST **transaminase** **glycolysis** enzyme **modulator**

IT Inflammation
(Crohn's disease, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Intestine, disease
(Crohn's, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Kidney, disease
(Goodpasture's syndrome, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Animal cell line
(MCF-7; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Animal cell line
(Novikoff-Hepatic; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Disease, animal
(adipose tissue, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Autoimmune disease
(autoimmune thrombocytopenia, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Autoimmune disease
Inflammation
Thyroid gland, disease
(autoimmune thyroiditis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Infection
(bacterial, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Human
(cells and enzymes; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Arthritis
(chronic, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Cartilage, disease
(degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)

IT Platelet (blood)
(disease, autoimmune thrombocytopenia, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for

treatment of different diseases)
IT Joint, anatomical
(disease, degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Adipose tissue
(disease, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Drugs
(**glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Intestine, disease
(inflammatory, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Cell proliferation
(inhibition; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Drug delivery systems
(injections, i.v.; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Autoimmune disease
(insulin-dependent diabetes mellitus, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Diabetes mellitus
(insulin-dependent, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Disease, animal
(joint degeneration, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT **Glycolysis**
(**modulation** of; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Drug delivery systems
(oral; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Arthritis
(polyarthritis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Asthma
Autoimmune disease
Cachexia
Connective tissue, disease
Diabetes insipidus
Multiple sclerosis
Myasthenia gravis
Psoriasis
Sepsis
(use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of different diseases)
IT Eye, disease
Inflammation
(uveitis, use to treat; **glycolysis** and/or **transaminase** complex **modulators** for treatment of

different diseases)
IT **9031-66-7, Transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(complex; **glycolysis** and/or **transaminase** complex
modulators for treatment of different diseases)
IT **9000-97-9**, Glutamate oxaloacetate **transaminase**
9001-47-2, Glutaminase **9001-59-6**, Pyruvate kinase
9001-64-3, Malate dehydrogenase **9014-27-1**, Serine
dehydratase **9015-68-3**, Asparaginase **9026-51-1**,
Nucleotide diphosphate kinase **9032-62-6**, Phosphoglyceromutase
9067-84-9, Deaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**glycolysis** and/or **transaminase** complex
modulators for treatment of different diseases)
IT **9031-66-7, Transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(complex; **glycolysis** and/or **transaminase** complex
modulators for treatment of different diseases)
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **9000-97-9**, Glutamate oxaloacetate **transaminase**
9001-47-2, Glutaminase **9001-59-6**, Pyruvate kinase
9001-64-3, Malate dehydrogenase **9014-27-1**, Serine
dehydratase **9015-68-3**, Asparaginase **9026-51-1**,
Nucleotide diphosphate kinase **9032-62-6**, Phosphoglyceromutase
9067-84-9, Deaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**glycolysis** and/or **transaminase** complex
modulators for treatment of different diseases)
RN 9000-97-9 HCAPLUS
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-47-2 HCAPLUS
CN Glutaminase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9014-27-1 HCAPLUS
CN Dehydratase, L-serine (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9015-68-3 HCAPLUS
CN Asparaginase (8CI, 9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9026-51-1 HCAPLUS
CN Kinase (phosphorylating), nucleoside diphosphate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9032-62-6 HCAPLUS

CN Phosphomutase, glycerate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9067-84-9 HCAPLUS
CN Deaminase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 7 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:3583 HCAPLUS
DN 140:71036
TI Modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof
IN Monia, Brett P.; Bennett, C. Frank; Baker, Brenda F.; Vickers, Timothy
PA USA
SO U.S. Pat. Appl. Publ., 54 pp., Cont.-in-part of U.S. Ser. No. 878,582, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004002153	A1	20040101	US 2003-336213	20030103
	US 6020199	A	20000201	US 1999-358381	19990721
	WO 2001007457	A1	20010201	WO 1999-US29594	19991214
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6284538	B1	20010904	US 2000-577902	20000524
	US 2002058638	A1	20020516	US 2001-878582	20010611
	WO 2004027030	A2	20040401	WO 2003-US29294	20030918
	WO 2004027030	A3	20050113		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2004137471	A1	20040715	US 2003-664639	20030918
	EP 1546344	A2	20050629	EP 2003-755836	20030918
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	WO 2004063329	A2	20040729	WO 2003-US41492	20031230
	WO 2004063329	A3	20050428		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO,				

NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ,
TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-358381 A1 19990721
WO 1999-US29594 A2 19991214
US 2000-577902 A1 20000524
US 2001-878582 B2 20010611
US 2002-411780P P 20020918
US 2003-336213 A 20030103
WO 2003-US29294 W 20030918

AB Oligomeric compds., compns. and methods are provided for modulating the expression of dual-specificity protein phosphatase PTEN. The compns. comprise oligomeric compds., particularly double stranded oligomeric compds., targeted to nucleic acids encoding PTEN. Specifically, a series of 18-nucleotide, phosphorothioate-linked oligonucleotides targeting the 5'-UTR, the coding region, or the 3'-UTR of dual-specificity protein phosphatase PTEN mRNA were synthesized. In transfected mammalian cells, 30 of 40 phosphorothioate-linked antisense oligonucleotides demonstrated at least 30% inhibition of PTEN gene expression. In addition, enhanced gene expression inhibition is observed from 18-nucleotide chimeric oligonucleotides derived from above phosphorothioate-linked antisense oligonucleotides, which is composed of a central gap region consisting of ten 2'-deoxynucleotides, which is flanked on both sides (5' and 3' directions) by four-nucleotide wings containing 2'-methoxyethyl (2'-MOE)nucleotides. Furthermore, a series of 21 nucleotide dsRNAs, formed by two oligonucleotides designed to contain the above 18 nucleobase oligonucleotides with one addnl. complementary base on the 3' end of the oligoribonucleotides followed by a two-nucleobase overhang of deoxythymidine (T), TT, also show PTEN inhibitory effect. A comparison of the inhibition of PTEN expression by single stranded oligonucleotides vs. double stranded RNA (dsRNA) is provided. Methods of using these compds. for modulation of PTEN expression and for treatment of diseases and conditions associated with expression of PTEN are provided. Such conditions include diabetes and hyperproliferative conditions. Methods for decreasing blood glucose levels, inhibiting PEPCK expression, decreasing blood insulin levels, decreasing insulin resistance, increasing insulin sensitivity, decreasing blood triglyceride levels or decreasing blood cholesterol levels in an animal, among others, using the compds. of the invention are also provided. The animal is preferably a human; also preferably the animal is a diabetic animal.

IC ICM A61K048-00
ICS C07H021-04; C12N005-00
INCL 435375000; 514044000; 536023200
CC 1-10 (**Pharmacology**)
Section cross-reference(s): 3, 7, **63**
ST antisense oligonucleotide dual specificity protein phosphatase PTEN inhibition therapy; dsRNA dual specificity protein phosphatase PTEN inhibition therapy
IT Genetic element
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(5'-untranslated region, of PTEN gene, antisense oligonucleotide or dsRNA targeted to; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)
IT Diabetes mellitus
(PTEN inhibition for the treatment of; modulation of dual-specificity

protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Adipose tissue
Human
Kidney
Liver
Mus
Rodentia
(PTEN inhibition in; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Gene, animal
RL: BSU (Biological study, unclassified); PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(PTEN; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Glycerides, analysis
RL: ANT (Analyte); ANST (Analytical study)
(blood, PTEN inhibition for the treatment of; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Drug delivery systems
(carriers, colloidal dispersion system; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Mutation
(deletion, PTEN dsRNA containing; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Metabolism, animal
(disorder, PTEN inhibition for the treatment of; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Body weight
(effect of PTEN inhibition on; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Genetic element
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(exon, of PTEN gene, antisense oligonucleotide or dsRNA targeted to; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Genetic vectors
(for PTEN dsRNA expression; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT cDNA sequences
(for human PTEN; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Post-transcriptional processing
(interference; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Genetic element
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)

(intron, of PTEN gene, antisense oligonucleotide or dsRNA targeted to; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Genetic element
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(intron/exon boundary, of PTEN gene, antisense oligonucleotide or dsRNA targeted to; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Antidiabetic agents
Blood analysis
Disease models
Drug delivery systems
Human
Rattus
(modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Antisense oligonucleotides
Phosphorothioate oligonucleotides
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Diabetes mellitus
(non-insulin-dependent, PTEN inhibition for the treatment of; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Protein sequences
(of human PTEN; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Double stranded RNA
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(small interfering, effect of PTEN inhibition on; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Mutation
(substitution, PTEN dsRNA containing; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT Kidney
Liver
(toxicity, PTEN inhibition in; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 256631-53-5P, ISIS 29576 256631-55-7P, ISIS 29578 256631-59-1P, ISIS 29582 256631-60-4P, ISIS 29583 256631-61-5P, ISIS 29584
256631-62-6P, ISIS 29585 256631-64-8P, ISIS 29587 256631-65-9P, ISIS 29588 256631-66-0P, ISIS 29589 256631-68-2P, ISIS 29591
256631-69-3P, ISIS 29592 256631-70-6P, ISIS 29593 256631-74-0P, ISIS 29597 256631-79-5P, ISIS 29602 256631-80-8P, ISIS 29603
256631-81-9P, ISIS 29604 256631-85-3P, ISIS 29608 256631-87-5P, ISIS

29610 256631-90-0P, ISIS 29613 256917-02-9P, ISIS 29535
256917-03-0P, ISIS 29536 256917-04-1P, ISIS 29537 256917-05-2P, ISIS
29538 256917-06-3P, ISIS 29539 256917-07-4P, ISIS 29540
256917-08-5P, ISIS 29541 256917-09-6P, ISIS 29542 256917-10-9P, ISIS
29543 256917-11-0P, ISIS 29544 256917-13-2P, ISIS 29546
256917-18-7P, ISIS 29551 256917-19-8P, ISIS 29552 256917-20-1P, ISIS
29553 256917-21-2P, ISIS 29554 256917-22-3P, ISIS 29555
256917-23-4P, ISIS 29556 256917-24-5P, ISIS 29557 256917-26-7P, ISIS
29559 256917-27-8P, ISIS 29560 256917-28-9P, ISIS 29561
256917-29-0P, ISIS 29562 256917-31-4P, ISIS 29564 256917-33-6P, ISIS
29566 256917-34-7P, ISIS 29567 256917-36-9P, ISIS 29569
256917-38-1P, ISIS 29571 256917-39-2P, ISIS 29572 256917-40-5P, ISIS
29573 357676-42-7P, ISIS 116847 357676-43-8P, ISIS 116845
634221-35-5P, ISIS 29581 634221-36-6P, ISIS 29590 640308-09-4P, ISIS
29534 640804-85-9P, ISIS 29545 640804-86-0P, ISIS 29547
640804-87-1P, ISIS 29548 640804-88-2P, ISIS 29549 640804-89-3P, ISIS
29568 640804-90-6P, ISIS 29570
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(PTEN antisense or dsRNA formation oligonucleotide; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 50-99-7, D-Glucose, analysis
RL: ANT (Analyte); ANST (Analytical study)
(blood, effect of PTEN inhibition on; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 9000-86-6, Alanine **transaminase 9000-97-9**, AST
37341-55-2, Phosphoenolpyruvatecarboxykinase
RL: ANT (Analyte); ANST (Analytical study)
(effect of PTEN inhibition on; **modulation** of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 149885-84-7, Dual-specificity protein phosphatase
RL: BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gene PTEN; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 391836-77-4, GenBank U92436 391836-79-6, GenBank U93051
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 640804-83-7 640804-84-8
RL: BSU (Biological study, unclassified); PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(nucleotide sequence; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 57-88-5, Cholest-5-en-3-ol (3β)-, analysis 9004-10-8, Insulin, analysis
RL: ANT (Analyte); ANST (Analytical study)
(serum, effect of PTEN inhibition on; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT 640806-64-0 640806-65-1 640806-66-2 640806-67-3 640806-68-4
640806-69-5 640806-70-8 640806-71-9 640806-72-0 640806-73-1

640806-74-2 640806-75-3 640806-76-4 640806-77-5 640806-78-6
640806-79-7 640806-80-0 640806-81-1 640806-82-2 640806-83-3
640806-84-4 640806-85-5 640806-86-6 640806-87-7 640806-88-8
640806-89-9 640813-29-2 640813-30-5 640813-31-6 640813-32-7
640813-33-8 640813-34-9 640813-35-0 640813-36-1 640813-37-2
640813-38-3 640813-39-4 640813-40-7 640813-41-8 640813-42-9
640813-43-0 640813-44-1 640813-45-2 640813-46-3 640813-47-4
640813-48-5 640813-49-6 640813-50-9 640813-51-0 640813-52-1
640813-53-2 640813-54-3 640813-55-4 640813-56-5 640813-57-6
640813-58-7 640813-59-8 640813-60-1 640813-61-2 640813-62-3
640813-63-4 640813-64-5 640813-65-6 640813-66-7 640813-67-8
640813-68-9 640813-69-0 640813-70-3 640813-71-4 640813-72-5
640813-73-6 640813-74-7 640813-75-8 640813-76-9 640813-77-0
640813-78-1 640813-79-2 640813-80-5 640813-81-6 640813-82-7
640813-83-8 640813-84-9 640813-85-0 640813-86-1 641650-92-2
RL: PRP (Properties)
(unclaimed nucleotide sequence; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

IT **9000-97-9**, AST
RL: ANT (Analyte); ANST (Analytical study)
(effect of PTEN inhibition on; modulation of dual-specificity protein phosphatase PTEN expression via antisense oligonucleotides and double-stranded RNAs and therapeutic use thereof)

RN 9000-97-9 HCAPLUS
CN Aminotransferase, aspartate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 8 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:142269 HCAPLUS
DN 139:80419
TI Modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes. II. Aromatic aldehydes
AU Niknahad, Hossein; Shuhendler, Adam; Galati, Giuseppe; Siraki, Arno G.; Easson, Elaine; Poon, Raymond; O'Brien, Peter J.
CS Department of Pharmacology and Toxicology, Faculty of Pharmacy, Shiraz University of Medical Sciences, Shiraz, 71345, Iran
SO Chemico-Biological Interactions (2003), 143-144, 119-128
CODEN: CBINA8; ISSN: 0009-2797
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
AB The mol. cytotoxic mechanisms of dietary benzaldehydes towards hepatocytes and its modulation by metabolizing enzymes were compared. Salicylaldehyde was found to be the most cytotoxic followed by cinnamaldehyde and both rapidly depleted some glutathione before an inhibition of respiration occurred, which preceded cell lysis. Reactive oxygen species were formed, but lipid peroxidn. was induced with cinnamaldehyde, but not salicylaldehyde. Glutathione depleted hepatocytes were more susceptible to cytotoxicity. Mitochondrial toxicity and cytotoxicity were prevented by glycolytic substrates (e.g. fructose), citric acid cycle substrates (e.g. glutamine) or cyclosporin, the mitochondrial permeability transition inhibitor. Inhibition of mitochondrial ALDH with chloral hydrate, crotonaldehyde or citral or decreasing mitochondrial NAD+ with rotenone increased cinnamaldehyde induced cytotoxicity with a much smaller effect on salicylaldehyde induced cytotoxicity. Cyanamide was the most effective ALDH inhibitor for increasing cinnamaldehyde induced cytotoxicity, presumably because cyanamide also inhibits microsomal ALDH. Although cinnamaldehyde was a better substrate than salicylaldehyde for ADH1,

cytosolic NADH generators (e.g. xylitol) prevented salicylaldehyde and cinnamaldehyde cytotoxicity similarly. This could be explained as salicylaldehyde was not a substrate for the ALDHs and would then be more dependent on ADH for detoxification.

CC 4-3 (Toxicology)

ST carbonyl cytotoxicity rat hepatocyte metabolizing enzyme arom aldehyde

IT Aldehydes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(aromatic; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Redox reaction
(biochem.; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Detoxification
(biol.; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(carbonyl-metabolizing; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Cytoplasm
(cytosol; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Liver
(hepatocyte; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Peroxidation
(lipid; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Cytolysis
Cytotoxicity
Glycolysis
Hepatotoxicity
Liver
Microsome
Mitochondria
Oxidative stress, biological
Rattus
Respiration, animal
Tricarboxylic acid cycle
(**modulating** carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Carbonyl complexes
Reactive oxygen species
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Biological transport
(permeation; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT Liver
(toxicity; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT 9028-86-8, Aldehyde dehydrogenase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(mitochondrial; modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT 90-02-8, Salicylaldehyde, biological studies 100-52-7, Benzaldehyde,

biological studies 100-83-4, 3-Hydroxy benzaldehyde 104-55-2, Cinnamaldehyde 121-33-5, Vanillin 123-08-0, 4-Hydroxy benzaldehyde 302-17-0, Chloral hydrate 4170-30-3, Crotonaldehyde 5392-40-5, Citral 7782-44-7D, Oxygen, reactive species
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT 420-04-2, Cyanamide
RL: ADV (Adverse effect, including toxicity); ARG (Analytical reagent use); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT 79217-60-0, Cyclosporin
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

IT 53-57-6, NADPH 53-84-9, NAD+ 56-85-9, L-Glutamine, biological studies 57-48-7, D-Fructose, biological studies 58-68-4, NADH 70-18-8, Glutathione, biological studies 87-99-0, Xylitol 9031-72-5, Alcohol dehydrogenase 27025-41-8, GSSG
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(modulating carbonyl cytotoxicity in intact rat hepatocytes by inhibiting carbonyl metabolizing enzymes and aromatic aldehydes)

RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 9 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:65461 HCAPLUS
DN 139:240134
TI Isoflurane Alters Energy Substrate Metabolism to Preserve Mechanical Function in Isolated Rat Hearts following Prolonged No-Flow Hypothermic Storage
AU Finegan, Barry A.; Gandhi, Manoj; Cohen, Matthew R.; Legatt, Donald; Clanachan, Alexander S.
CS Departments of Anesthesiology and Pain Medicine, Univ. Alberta, Edmonton, AB, T6G 2B7, Can.
SO Anesthesiology (2003), 98(2), 379-386
CODEN: ANESAV; ISSN: 0003-3022
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB BACKGROUND Isoflurane enhances mech. function in hearts subject to normothermic global or regional ischemia. The authors examined the effectiveness of isoflurane in preserving mech. function in hearts subjected to cardioplegic arrest and prolonged hypothermic no-flow storage. The role of isoflurane in altering myocardial glucose metabolism during storage and reperfusion during these conditions and the contribution of ATP-sensitive potassium (KATP) channel activation in mediating the functional and metabolic effects of isoflurane preconditioning was determined METHODS Isolated working rat hearts were subjected to cardioplegic arrest with St. Thomas' II solution, hypothermic no-flow storage for 8 h, and subsequent aerobic reperfusion. The consequences of isoflurane treatment were assessed during the following conditions: (1) isoflurane exposure before and during storage; (2) brief isoflurane exposure during early nonworking poststorage reperfusion; and (3) isoflurane preconditioning before storage. The selective mitochondrial and sarcolemmal KATP channel antagonists, 5-hydroxydecanoate

and HMR 1098, resp., were used to assess the role of KATP channel activation on glycogen consumption during storage in isoflurane-preconditioned hearts. RESULTS Isoflurane enhanced recovery of mech. function if present before and during storage. Isoflurane preconditioning was also protective. Isoflurane reduced glycogen consumption during storage under the aforementioned circumstances. Storage of isoflurane-preconditioned hearts in the presence of 5-hydroxydecanoate prevented the reduction in glycogen consumption during storage and abolished the beneficial effect of isoflurane preconditioning on recovery of mech. function. CONCLUSIONS Isoflurane provides additive protection of hearts subject to cardioplegic arrest and prolonged hypothermic no-flow storage and favorably alters energy substrate metabolism by **modulating glycolysis** during ischemia. The effects of isoflurane preconditioning on glycolysis during hypothermic no-flow storage appears to be associated with activation of mitochondrial KATP channels.

CC 1-11 (**Pharmacology**)
Section cross-reference(s): 13

ST isoflurane heart energy substrate metab preservation function

IT Potassium channel
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ATP-dependent; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT Heart
(cardioplegia; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT Cytoprotective agents
(cardioprotective; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT Heart
Hypothermia
Organ preservation
(isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT Heart
(toxicity; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT 26675-46-7, Isoflurane
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT 50-99-7, D-Glucose, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(metabolism; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

IT 9005-79-2, Glycogen, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reduced consumption; isoflurane alters energy substrate metabolism to preserve mech. function in isolated rat hearts following prolonged No-flow hypothermic storage)

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 10 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:792041 HCAPLUS

DN 137:289048
TI Use of sugar phosphates, sugar phosphate analogs, amino acids and/or amino acid analogs for the **modulation** of the **glycolysis** enzyme complex of the malate aspartate shuttle and/or transaminases, and therapeutic use
IN Eigenbrodt, Erich; Mazurek, Sybille; Grimm, Helmut
PA ScheBo Biotech AG, Germany
SO Ger. Offen., 14 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 10164711	A1	20021017	DE 2001-10164711	20010313
PRAI DE 2001-10164711		20010313		

OS MARPAT 137:289048
AB The invention discloses the use of amino acids, amino acid analogs, sugar phosphates, sugar phosphate-analogs, and mixts. of such substances, for the production of a pharmaceutical composition for the treatment of tumors and/or for immunosuppression and/or the treatment of sepsis by **modulation** of the association of the **glycolysis** enzyme complex/M2-PK and/or by inhibition of transaminases and/or by dissociation of the bond of malate dehydrogenase at p36.
IC ICM A61K031-198
CC 1-12 (**Pharmacology**)
ST amino acid sugar phosphate antitumor immunosuppressant; sepsis treatment amino acid sugar phosphate; **glycolysis** enzyme complex **modulation** amino acid sugar phosphate; malate dehydrogenase **transaminase modulation** amino acid sugar phosphate
IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(glycolytic; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)
IT Carcinoma
(hepatocellular; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)
IT Liver, neoplasm
(hepatoma; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)
IT Drug delivery systems
(injections, i.v.; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(membrane, p36; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)
IT Antitumor agents
Immunosuppressants
Neoplasm
Sepsis
(sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT Amino acids, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT Carbohydrates, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sugar phosphates; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT **9001-59-6**, Pyruvate kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M2-PK; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT 52-90-4, L-Cysteine, biological studies 52-90-4D, L-Cysteine, analogs 56-45-1, L-Serine, biological studies 56-45-1D, L-Serine, analogs 61-90-5, L-Leucine, biological studies 61-90-5D, L-Leucine, analogs 62-57-7, Aminoisobutyric acid 62-57-7D, Aminoisobutyric acid, analogs 63-68-3, L-Methionine, biological studies 63-68-3D, L-Methionine, analogs 68-41-7, Cycloserine 68-41-7D, Cycloserine, analogs 72-18-4, L-Valine, biological studies 72-18-4D, L-Valine, analogs 73-32-5, L-Isoleucine, biological studies 73-32-5D, L-Isoleucine, analogs 138-81-8, Glycerate 2,3-diphosphate 138-81-8D, Glycerate 2,3-diphosphate, analogs 147-85-3, L-Proline, biological studies 147-85-3D, L-Proline, analogs 488-69-7, Fructose-1,6-bisphosphate 488-69-7D, Fructose-1,6-bisphosphate, analogs 645-88-5, Aminooxyacetic acid 645-88-5D, Aminooxyacetic acid, analogs 820-11-1 820-11-1D, analogs 14689-84-0, Ribose-1,5-diphosphate 14689-84-0D, Ribose-1,5-diphosphate, analogs 24218-00-6, Ribulose-1,5-bisphosphate 24218-00-6D, Ribulose-1,5-bisphosphate, analogs 108605-62-5 108605-62-5D, analogs
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

IT **9001-59-6**, Pyruvate kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M2-PK; sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(sugar phosphates, amino acids, and analogs for **modulation** of **glycolysis** enzyme complex of malate aspartate shuttle and/or transaminases, and therapeutic use)

RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 11 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:716073 HCAPLUS
DN 137:226595
TI Use of sugar phosphates, sugar phosphate analogues, amino acids, amino acid analoges for **modulating transaminases** and/or the association of p36/malate dehydrogenase
IN Eigenbrodt, Erich; Mazurek, Sybille; Muellner, Stefan
PA Germany
SO PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002072078	A2	20020919	WO 2002-DE921	20020312
	WO 2002072078	A3	20021212		
	WO 2002072078	C2	20030130		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10112925	A1	20021002	DE 2001-10112925	20010313
	EP 1372646	A2	20040102	EP 2002-750522	20020312
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2005054584	A1	20050310	US 2004-471866	20040816
PRAI	DE 2001-10112925	A	20010313		
	WO 2002-DE921	W	20020312		

OS MARPAT 137:226595
AB The invention relates to the use of a substance selected from the group consisting of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analogs and mixts. of said substances, for producing a pharmaceutical composition for reducing weight and/or preventing delayed damage caused by diabetes mellitus by modulating the association p36/malate dehydrogenase and/or transaminases. The substances are used in i.v. formulations and as food supplements along with insulin administration.
IC ICM A61K031-00
CC 1-6 (**Pharmacology**)
Section cross-reference(s): 17, **63**
ST sugar phosphate amino acid diabetes mellitus
IT Drug delivery systems
(injections, i.v.; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(membrane, p36; use of sugar phosphates, sugar phosphate analogs, amino

acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Glycopeptides
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(serine-containing; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Carbohydrates, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sugar phosphates; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Diet
(supplements; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Drug delivery systems
(tablets; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Diabetes mellitus
(use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT Amino acids, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT 52-90-4, L-Cysteine, biological studies 56-45-1, L-Serine, biological studies 61-90-5, L-Leucine, biological studies 62-57-7 63-68-3, L-Methionine, biological studies 72-18-4, L-Valine, biological studies 73-32-5, L-Isoleucine, biological studies 138-81-8, Glycerate-2,3-diphosphate 147-85-3, L-Proline, biological studies 488-69-7 645-88-5 2002-28-0, Ribulose-1,5-diphosphate 9004-10-8, Insulin, biological studies 14689-84-0, Ribose-1,5-diphosphate 108605-62-5
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of sugar phosphates, sugar phosphate analogs, amino acids, amino acid analoges for **modulating transaminases** and association of p36/malate dehydrogenase)

RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 12 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:716072 HCAPLUS
DN 137:226594
TI Use of sugar phosphate, sugar phosphate analogues, amino acids and/or amino acid analogues for **modulating** the **glycolysis**-enzyme complex, the malate-aspartate shuttle and/or transaminases
IN Eigenbrodt, Erich; Mazurek, Sybille; Grimm, Helmut
PA Schebo Biotech A.-G., Germany
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002072077	A2	20020919	WO 2002-DE212	20020117
	WO 2002072077	A3	20021227		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10112926	A1	20021002	DE 2001-10112926	20010313
	EP 1368018	A2	20031210	EP 2002-704608	20020117
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004524326	T2	20040812	JP 2002-571036	20020117
	US 2004235755	A1	20041125	US 2004-471705	20040604
PRAI	DE 2001-10112926	A	20010313		
	WO 2002-DE212	W	20020117		

OS MARPAT 137:226594
AB The invention relates to the use of a substance selected from the group consisting of "amino acids, amino acid analogs, sugar phosphates, sugar phosphate analogs and mixts. of substances of this type" for producing a pharmaceutical composition for treating tumors and/or for immunosuppression and/or sepsis by **modulating** the association of the **glycolysis**-enzyme complex/M2-PK and/or by inhibiting transaminases and/or by dissolving the malate dehydrogenase bond with p36.
IC ICM A61K031-00
CC 1-6 (**Pharmacology**)
Section cross-reference(s): 7, **63**
ST sugar phosphate amino acid neoplasm immunosuppression
IT Drug delivery systems
(injections, i.v.; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating** **glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(membrane, p36; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating** **glycolysis** -enzyme complex, malate-aspartate shuttle and transaminases)
IT Glycopeptides

RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(serine-containing; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT Carbohydrates, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sugar phosphates; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT Immunosuppression
Neoplasm
Sepsis
(use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT Amino acids, biological studies
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT **9001-59-6**, Pyruvate kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M2 isoenzyme, inhibitors of; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT 52-90-4, L-Cysteine, biological studies 56-45-1, L-Serine, biological studies 61-90-5, L-Leucine, biological studies 62-57-7 63-68-3, L-Methionine, biological studies 72-18-4, L-Valine, biological studies 73-32-5, L-Isoleucine, biological studies 138-81-8, Glycerate-2,3-diphosphate 147-85-3, L-Proline, biological studies 488-69-7 645-88-5 2002-28-0, Ribulose-1,5-diphosphate 14689-84-0, Ribose-1,5-diphosphate 108605-62-5
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

IT **9001-59-6**, Pyruvate kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M2 isoenzyme, inhibitors of; use of sugar phosphate, sugar phosphate analogs, amino acids, amino acid analogs for **modulating glycolysis**-enzyme complex, malate-aspartate shuttle and transaminases)

RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **9001-64-3**, Malate dehydrogenase **9031-66-7**, Transaminase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors of; use of sugar phosphate, sugar phosphate analogs, amino

acids, amino acid analogs for **modulating glycolysis**
-enzyme complex, malate-aspartate shuttle and transaminases)
RN 9001-64-3 HCAPLUS
CN Dehydrogenase, malate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 9031-66-7 HCAPLUS
CN Aminotransferase (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 13 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:905538 HCAPLUS
DN 136:177621
TI Adenine nucleotide translocator mediates the mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437
AU Belzacq, Anne-Sophie; El Hamel, Chahrazed; Vieira, Helena L. A.; Cohen, Isabel; Haouzi, Delphine; Metivier, Didier; Marchetti, Philippe; Brenner, Catherine; Kroemer, Guido
CS CNRS-UM R6022, Universite de Technologie de Compiegne, Compiegne, F-60205, Fr.
SO Oncogene (2001), 20(52), 7579-7587
CODEN: ONCNES; ISSN: 0950-9232
PB Nature Publishing Group
DT Journal
LA English
AB An increasing number of exptl. chemotherapeutic agents induce apoptosis by directly triggering mitochondrial membrane permeabilization (MMP). Here we examined MMP induced by lonidamine, arsenite, and the retinoid derivative CD437. Cells overexpressing the cytomegalovirus-encoded protein vMIA, a protein which interacts with the adenine nucleotide translocator, were strongly protected against the MMP-inducing and apoptogenic effects of lonidamine, arsenite, and CD437. In a cell-free system, lonidamine, arsenite, and CD437 induced the permeabilization of ANT proteoliposomes, yet had no effect on protein-free liposomes. The ANT-dependent membrane permeabilization was inhibited by the two ANT ligands ATP and ADP, as well as by recombinant Bcl-2 protein. Lonidamine, arsenite, and CD437, added to synthetic planar lipid bilayers containing ANT, elicited ANT channel activities with clearly distinct conductance levels of 20±7, 100±30, and 47±7 pS, resp. Altering the ATP/ADP gradient built up on the inner mitochondrial membrane by inhibition of **glycolysis** and/or oxidative phosphorylation differentially **modulated** the cytocidal potential of lonidamine, arsenite, and CD437. Inhibition of F0F1ATPase without glycolysis inhibition sensitized to lonidamine-induced cell death. In contrast, only the combined inhibition of glycolysis plus F0F1ATPase sensitized to arsenite-induced cell death. No sensitization to cell death induction by CD437 was achieved by glucose depletion and/or oligomycin addition These results indicate that ANT is a target of lonidamine, arsenite, and CD437 and unravel an unexpected heterogeneity in the mode of action of these three compds.
CC 1-6 (**Pharmacology**)
ST adenine nucleotide translocator mitochondria membrane permeability lonidamine arsenite CD437
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ADP/ATP carrier; adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)
IT Apoptosis
Glycolysis

Liposomes
Oxidative phosphorylation, biological
(adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

IT Ion channel
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

IT Mitochondria
(membrane; adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

IT Membrane, biological
(mitochondrial; adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

IT Biological transport
(permeation; adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

IT 15502-74-6, Arsenite 50264-69-2, Lonidamine 125316-60-1, CD437
RL: PAC (Pharmacological activity); BIOL (Biological study)
(adenine nucleotide translocator mediates mitochondrial membrane permeabilization induced by lonidamine, arsenite and CD437)

RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 14 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:886508 HCAPLUS
DN 136:31642
TI Modulators of hr44 as therapeutic compounds
IN Braun, Gabriele; Mckechnie, Nicol
PA University of Bristol, UK
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001092521	A1	20011206	WO 2001-GB2397	20010530
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2409191	AA	20011206	CA 2001-2409191	20010530
	EP 1305413	A1	20030502	EP 2001-934163	20010530
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2003134816	A1	20030717	US 2002-300575	20021120
PRAI	GB 2000-13105	A	20000530		
	WO 2001-GB2397	W	20010530		

AB The present invention relates to a compound capable of modulating the activity and/or expression of hr44, for use in therapy. Drug screening methods are also disclosed.

IC ICM C12N015-12
ICS C07K014-47; G01N033-53; C07K016-18; C07K016-30; A61K039-395

CC 1-1 (**Pharmacology**)

Section cross-reference(s): 9, 15, **63**
ST drug screening hr44 sequence antitumor diagnostic
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(YP4; modulators of hr44 as therapeutic compds.)
IT Diagnosis
(agents; modulators of hr44 as therapeutic compds.)
IT Tumor antigens
Tumor antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antibodies to; modulators of hr44 as therapeutic compds.)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(fibrillarins; modulators of hr44 as therapeutic compds.)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hnRNPE1; modulators of hr44 as therapeutic compds.)
IT Proteins
RL: BSU (Biological study, unclassified); PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(hr44; modulators of hr44 as therapeutic compds.)
IT Fatty acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(metabolism of; modulators of hr44 as therapeutic compds.)
IT Antitumor agents
Blood vessel, disease
Diagnosis
Drug delivery systems
Drug screening
Glycolysis
Human
Neoplasm
Protein sequences
RNA splicing
cDNA sequences
(**modulators** of hr44 as therapeutic compds.)
IT Prostaglandins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(modulators of hr44 as therapeutic compds.)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(modulators of hr44 as therapeutic compds.)
IT Biological transport
(of fatty acids; modulators of hr44 as therapeutic compds.)
IT 379739-29-4
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; modulators of hr44 as therapeutic compds.)
IT **9001-59-6**, Pyruvate kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(modulators of hr44 as therapeutic compds.)
IT 379739-28-3, DNA (human protein YP4 cDNA plus flanks)
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; modulators of hr44 as therapeutic compds.)
IT 379693-36-4 379693-37-5 379693-38-6 379693-39-7 380227-77-0
RL: PRP (Properties)
(unclaimed sequence; modulators of hr44 as therapeutic compds.)
IT **9001-59-6**, Pyruvate kinase

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(modulators of hr44 as therapeutic compds.)
RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 15 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:309344 HCAPLUS
DN 135:102272
TI Modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C
AU Schafer, M.; Bahde, D.; Bosche, B.; Ladilov, Y.; Schafer, C.; Piper, H. M.; Noll, T.
CS Physiologisches Institut, Justus-Liebig-Universitat, Giessen, D-35392, Germany
SO American Journal of Physiology (2001), 280(3, Pt. 2), H1002-H1010
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
AB When energy metabolism is disrupted, endothelial cells lose Ca2+ from endoplasmic reticulum (ER) and the cytosolic Ca2+ concentration ([Ca2+]i) increases. The importance of glycolytic energy production and the mechanism of Ca2+ loss from the ER were analyzed. Endothelial cells from porcine aorta in culture and in situ were used as models. 2-Deoxy-D-glucose (2-DG, 10 mM), an inhibitor of glycolysis, caused an increase in [Ca2+]i (measured with fura 2) within 1 min when total cellular ATP contents were not yet affected. Stimulation of oxidative energy production with pyruvate (5 mM) did not attenuate this 2-DG-induced rise of [Ca2+]i, while this maneuver preserved cellular ATP contents. The inhibitor of ER-Ca2+-ATPase, thapsigargin (10 nM), augmented the 2-DG-induced rise of [Ca2+]i. Xestospongin C (3 μM), an inhibitor of D-myo-inositol 3-phosphate [Ins(3)P]-sensitive ER-Ca2+ release, abolished the rise. The results demonstrate that the ER of endothelial cells is very sensitive to glycolytic metabolic inhibition. When this occurs, the ER Ca2+ store is discharged by opening of the Ins(3)P-sensitive release channel. Xestospongin C can effectively suppress the early [Ca2+]i rise in metabolically inhibited endothelial cells.
CC 1-8 (**Pharmacology**)
Section cross-reference(s): 13, 14
ST glycolysis aorta endothelium calcium xestospongin C
IT Calcium channel
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Ins(3)P-sensitive; modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT Artery
(aorta, endothelium; modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT Cytoplasm
(cytosol; modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT **Glycolysis**
(**modulation** of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT 9000-83-3
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

(Biological study); PROC (Process)
(calcium-dependent; modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT 14127-61-8, Ca2+, biological studies
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT 88903-69-9, Xestospongin C
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
IT 2831-74-5, D-myo-Inositol 3-phosphate
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of early [Ca2+]i rise in metabolically inhibited endothelial cells by xestospongin C)
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 16 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:164194 HCAPLUS
DN 135:14222
TI GABAergic blockade of cocaine-associated cue-induced increases in nucleus accumbens dopamine
AU Gerasimov, M. R.; Schiffer, W. K.; Gardner, E. L.; Marsteller, D. A.; Lennon, I. C.; Taylor, S. J. C.; Brodie, J. D.; Ashby, C. R.; Dewey, S. L.
CS Chemistry Department, Brookhaven National Laboratory, Upton, NY, 11973, USA
SO European Journal of Pharmacology (2001), 414(2/3), 205-209
CODEN: EJPHAZ; ISSN: 0014-2999
PB Elsevier Science B.V.
DT Journal
LA English
AB Environments previously associated with drug use can become one of the most common factors triggering relapse to drug-seeking behavior. To better understand the neurochem. mechanisms potentially mediating these cues, we measured nucleus accumbens dopamine levels in animals exposed to environmental cues previously paired with cocaine administration. In animals exposed to a cocaine-paired environment nucleus accumbens dopamine increased by 25%. When administered 2.5 h prior to presentation of the environmental trigger, racemic vigabatrin (an irreversible inhibitor of γ-aminobutyric acid (GABA)-transaminase) abolished this cue-induced increase. Conversely, R-(-)-vigabatrin, the inactive enantiomer, had no effect. Combined with our earlier findings, these studies support the potential therapeutic benefit of this enzyme-based GABAergic strategy to modulate brain dopamine and the subsequent treatment of drug addiction.
CC 1-11 (**Pharmacology**)
ST GABA transaminase brain dopamine cocaine addiction
IT Neurotransmission
(GABAergic; therapeutic benefit of GABAergic blockade to modulate brain dopamine in cocaine addiction)
IT Brain
(nucleus accumbens; therapeutic benefit of GABAergic blockade to modulate brain dopamine in cocaine addiction)
IT Drug dependence
(therapeutic benefit of GABAergic blockade to modulate brain dopamine

in cocaine addiction)
IT 50-36-2, Cocaine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(therapeutic benefit of GABAergic blockade to modulate brain dopamine in cocaine addiction)
IT 51-61-6, Dopamine, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutic benefit of GABAergic blockade to modulate brain dopamine in cocaine addiction)
IT 9037-67-6, GABA **transaminase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(therapeutic benefit of GABAergic blockade to **modulate** brain dopamine in cocaine addiction)
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 17 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:415156 HCAPLUS
DN 133:276145
TI GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice
AU Abel, Marc S.; Kohli, Neelu
CS Department of Cell Biology and Anatomy, FUHS/The Chicago Medical School, North Chicago, IL, 60064, USA
SO Metabolic Brain Disease (1999), 14(4), 253-263
CODEN: MBDIEE; ISSN: 0885-7490
PB Kluwer Academic/Plenum Publishers
DT Journal
LA English
AB The mechanism of action of many anticonvulsive agents is to increase the function of the GABAergic system. Inhibition of GABA-Transaminase (GABA-T), the degradative enzyme for GABA, increases GABA levels in the brain. In this study, antisense oligodeoxynucleotides (ASO) targeted at the start codon region of GABA-Transaminase mRNA were used to modify seizure activity. Mice were treated, by intracerebroventricular injection, with antisense oligos or appropriate controls. At various times after treatment, the animals were challenged with cocaine (70 mg/kg, i.p.) and observed for seizure activity. At 15 h after treatment, 1.152 and 1.44 nmol antisense oligo blocked cocaine-induced seizures. There was no effect of antisense oligo 8 or 36 h after treatment. In addition, treatment with 7.2 nmol antisense oligo prevented pentylenetetrazol-induced seizures. These data demonstrate the modulation of seizure threshold using antisense oligodeoxynucleotides to GABA-T.
CC 1-11 (**Pharmacology**)
ST GABA transaminase antisense oligodeoxynucleotide anticonvulsant
IT Anticonvulsants
GABA agonists
(GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice)
IT Antisense oligonucleotides
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice)
IT mRNA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(GABA-**transaminase**-specifying; GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice)

IT 300433-48-1 300433-49-2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice)

IT 9037-67-6, GABA-**transaminase**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(GABA-**transaminase** antisense oligodeoxynucleotide **modulates** cocaine- and pentylenetetrazol-induced seizures in mice)

RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 18 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:390180 HCAPLUS
DN 131:196547
TI Magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis
AU Ronen, S. M.; DiStefano, F.; McCoy, C. L.; Robertson, D.; Smith, T. A. D.; Al-Saffar, N. M.; Titley, J.; Cunningham, D. C.; Griffiths, J. R.; Leach, M. O.; Clarke, P. A.
CS Clinical Magnetic Resonance Research Group, Cancer Research Campaign (CRC), Sutton, Surrey, SM2 5PT, UK
SO British Journal of Cancer (1999), 80(7), 1035-1041
CODEN: BJCAAI; ISSN: 0007-0920
PB Churchill Livingstone
DT Journal
LA English
AB Apoptosis was induced by treating L1210 leukemia cells with mechlorethamine, and SW620 colorectal cells with doxorubicin. The onset and progression of apoptosis were monitored by assessing caspase activation, mitochondrial transmembrane potential, phosphatidylserine externalization, DNA fragmentation and cell morphol. In parallel, 31P magnetic resonance (MR) spectra of cell exts. were recorded. In L1210 cells, caspase activation was detected at 4 h. By 3 h, the MR spectra showed a steady decrease in NTP and NAD, and a significant build-up of fructose 1,6-bisphosphate (F-1,6-P,) dihydroxyacetonephosphate and glycerol-3-phosphate, indicating **modulation** of **glycolysis.** Treatment with iodoacetate also induced a build-up of F-1,6-P, while preincubation with two poly(ADP-ribose) polymerase inhibitors, 3-aminobenzamide and nicotinamide, prevented the drop in NAD and the build-up of glycolytic intermediates. This suggested that our results were due to inhibition of glyceraldehyde-3-phosphate dehydrogenase, possibly as a consequence of NAD depletion following poly(ADP-ribose) polymerase activation. Doxorubicin treatment of the adherent SW620 cells caused cells committed to apoptosis to detach. F-1,6-P was observed in detached cells, but not in treated cells that remained attached. This indicated that our observations were not cell line- or treatment-specific, but were correlated with the appearance of apoptotic cells following drug treatment. The 31P MR spectrum of tumors responding to chemotherapy could be modulated by similar effects.
CC 9-5 (Biochemical Methods)

Section cross-reference(s): 1
ST NMR spectroscopy cell chemotherapy apoptosis metab
IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(fragmentation; magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT Apoptosis
Cell morphology
Chemotherapy
Glycolysis
Leukemia
Metabolism, animal
Staining, biological
(magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT Nucleoside triphosphates
Phosphatidylserines
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT Animal tissue culture
(mammalian, colorectal cells and leukemia cells; magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT NMR spectroscopy
(phosphorus-31; magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT 51-75-2, Mechlorethamine 64-69-7, Acetic acid, iodo- 98-92-0, Nicotinamide 3544-24-9, 3-Aminobenzamide 23214-92-8, Doxorubicin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT 9055-67-8, Poly(ADP-ribose)polymerase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
IT 53-84-9, NAD 57-03-4, Glycerol-3-phosphate 57-04-5, Dihydroxyacetonephosphate 488-69-7, Fructose 1,6-bisphosphate
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(magnetic resonance detects metabolic changes associated with chemotherapy-induced apoptosis)
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 19 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:128775 HCAPLUS
DN 128:226162
TI Fructose-1,6-bisphosphate preserves adenosine triphosphate but not intracellular pH during hypoxia in respiring neonatal rat brain slices
AU Espanol, Maryceline T.; Litt, Lawrence; Hasegawa, Koh; Chang, Lee-Hong; Macdonald, Jeffrey M.; Gregory, George; James, Thomas L.; Chan, Pak H.
CS Departments of Anesthesia, Pharmaceutical Chemistry, Neurology, Neurosurgery, Radiology, The University of California, San Francisco, San Francisco, CA, 94143-0648, USA
SO Anesthesiology (1998), 88(2), 461-472
CODEN: ANESAV; ISSN: 0003-3022
PB Lippincott-Raven Publishers

DT Journal
LA English
AB Fructose-1,6-bisphosphate (FBP) sometimes provides substantial cerebral protection during hypoxia or ischemia. 31P/1H NMR spectroscopy of cerebrocortical slices was used to study the effects of FBP on hypoxia-induced metabolic changes. In addition, 13C-labeled glucose was administered and 13C NMR spectroscopy was used to search for FBP-induced **modulations** in **glycolysis** and the pentose-phosphate pathway. In each experiment, 80 slices (350 μm) obtained from ten 7-day-old Sprague-Dawley rat litter mates were placed together in a 20-mm NMR tube, perfused, and subjected to 30 min of hypoxia (P02 < 3 mmHg). Nine expts. were performed, with in each of three groups: (1) no treatment with FBP; (2) 60 min of prehypoxia treatment with FBP (2 mM); and (3) 60 min of posthypoxia treatment with FBP (2 mM). 31P/1H Interleaved NMR spectra at 4.7 T provided average ATP, intracellular pH, and lactate. Cresyl violet stains of random slices taken at predetd. time points were studied histol. Some expts. had [2-13C]glucose in the perfusate. Slices from these studies were frozen for perchloric acid extraction of intracellular metabolites and studied with high-resolution 13C NMR spectroscopy at 11.75 T. With no pretreatment with FBP, hypoxia caused an ≈50% loss of ATP, an ≈700% increase in lactate, and a decrease in intracellular pH to ≈6.4. Pretreatment with FBP resulted in no detectable loss of ATP, no increase in lactate, and minimal morphol. changes but did not alter decreases in intracellular pH. 13C NMR spectra of extracted metabolites showed that pretreatment caused accumulation of [1-13C]fructose-6-phosphate, an early pentose-phosphate pathway metabolite. Posthypoxic treatment with FBP had no effects compared with no treatment. During severe hypoxia, pretreatment with FBP completely preserves ATP and almost completely preserves cell morphol. but does not alter hypoxia-induced decreases in intracellular pH. Pretreatment also substantially augments the flux of glucose into the pentose phosphate pathway.
CC 1-11 (**Pharmacology**)
ST fructose bisphosphate ATP hypoxia brain
IT Brain
Energy metabolism, animal
Glycolysis
Hypoxia, animal
Pentose phosphate pathway
(fructose bisphosphate preserves ATP but not intracellular pH during cerebral hypoxia)
IT Cytoprotective agents
(neuroprotectants; fructose bisphosphate preserves ATP but not intracellular pH during cerebral hypoxia)
IT 488-69-7, Fructose-1,6-bisphosphate
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(fructose bisphosphate preserves ATP but not intracellular pH during cerebral hypoxia)
IT 56-65-5, 5'-ATP, biological studies 67-07-2, Phosphocreatine
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(fructose bisphosphate preserves ATP but not intracellular pH during cerebral hypoxia)
IT 50-21-5, Lactic acid, biological studies 643-13-0, Fructose-6-phosphate
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(fructose bisphosphate preserves ATP but not intracellular pH during cerebral hypoxia)
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD

KATHLEEN FULLER EIC 1700 REMSON 4B28 571/272-2505

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 20 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:644409 HCAPLUS
DN 127:326458
TI Dexfenfluramine modulates hepatic glycogen metabolism by a calcium-dependent pathway
AU Comte, Blandine; Romanelli, Angela; Haddad, Pierre; Van De Werve, Gerald
CS Laboratoire d'endocrinologie metabolique, Departement de nutrition, Universite de Montreal, Montreal, QC H3C 3J7, Can.
SO Canadian Journal of Physiology and Pharmacology (1997), 75(7), 842-848
CODEN: CJPPA3; ISSN: 0008-4212
PB National Research Council of Canada
DT Journal
LA English
AB In this study, the mechanism of action of dexfenfluramine (DEXF) at the hepatic level was investigated. The drug is shown to bind to the α1-adrenergic receptor and to increase intracellular calcium in isolated rat hepatocytes, thereby activating phosphorylase via a calcium-dependent mechanism. Moreover, phosphorylase activation by DEXF was inhibited by different agents that interfere with the α1-adrenergic signaling system: prazosin, phorbol 12α-myristate 13β- acetate (PMA), and DEXF itself. We also show that phosphorylase activation induced by catecholamines and analogs (epinephrine, phenylephrine), whose actions are mediated by a calcium-dependent mechanism, was counteracted by the drug in the submillimolar range (0.1-1 mM). The activation of glycogenolysis by the drug is accompanied by a stimulation of the glycolytic flux (54% increase in lactate plus pyruvate accumulation), consistent with an increase in fructose-2,6-bisphosphate (F-2,6-BP) levels (36%). These results indicate that the interaction of DEXF with the α1-adrenergic receptor channels glucose 6-phosphate derived from glycogen away from glucose production into the glycolytic pathway.
CC 1-12 (**Pharmacology**)
ST dexfenfluramine liver glycogen calcium phosphorylase; alpha1 adrenoceptor phosphorylase glycogen dexfenfluramine
IT **Glycolysis**
Liver
(dexfenfluramine **modulates** hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)
IT Catecholamines, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)
IT Liver
(hepatocyte; dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)
IT Adrenoceptor agonists
(α1-; dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)
IT Adrenoceptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(α1; dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to

α1-adrenergic receptor)

IT 9035-74-9, Phosphorylase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(activation of; dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)

IT 51-43-4, Epinephrine 59-42-7, Phenylephrine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)

IT 3239-44-9, Dexfenfluramine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)

IT 50-21-5, Lactic acid, biological studies 50-99-7, Glucose, biological studies 56-73-5, Glucose 6-phosphate 127-17-3, Pyruvic acid, biological studies 7440-70-2, Calcium, biological studies 9005-79-2, Glycogen, biological studies 77164-51-3, Fructose-2,6-bisphosphate
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)

IT 7440-70-2, Calcium, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(transport; dexfenfluramine modulates hepatic glycogen metabolism by calcium-dependent activation of phosphorylase and by binding to α1-adrenergic receptor)

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L63 ANSWER 21 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1996:642298 HCAPLUS
DN 125:321775
TI Effects of the inhibitors of energy metabolism, lonidamine and levamisole, on 5-aminolevulinic-acid-induced photochemotherapy
AU Shevchuk, Igor; Chekulayev, Vladimir; Moan, Johan; Berg, Kristian
CS Institute Chemistry, Estonian Academy Science, Tallinn, EE0026, Estonia
SO International Journal of Cancer (1996), 67(6), 791-799
CODEN: IJCNAW; ISSN: 0020-7136
PB Wiley-Liss
DT Journal
LA English
AB The ability of endogenously synthesized protoporphyrin IX (PpIX) to damage Chinese hamster lung fibroblasts of the line V79 by exposure to light was examined This treatment induced reduction of cellular ATP, GTP, of the NADH/NAD+ ratio and of oxygen consumption. The present results indicate a close relationship between inhibition of respiration of irradiated cells and their ability to survive, e.g., 1 min of light exposure induced 90% inhibition of oxygen consumption and inactivation of approx. 95% of the cells, while the cellular content of ATP was reduced by only 15%. This indicates that the mitochondria are one of the primary targets of 5-aminolevulinic acid (ALA)-mediated photochemotherapy (PCT). In the

present study, ALA-PCT was combined with the **modulators** of the **glycolysis** and the respiration chain, levamisole (LEV) and lonidamine (LND). A synergistic effect of combining ALA-PCT with non-toxic concns. of LND was observed when LND was given prior to light exposure. This synergism was observed despite a substantial LND-induced inhibition of PpIX formation. At increasing doses of LND (>0.15 mM) the combination treatment becomes less efficient. This is due to the inhibition of PpIX synthesis induced by LND. A synergistic effect of ALA-PDT and LEV was found when LEV was given prior to light exposure. This was at least partly due to an LEV-stimulated effect on ALA-induced PpIX formation. However, it is not clear from the present results whether LEV may perturb energy metabolism in V79 cells since LEV alone did not reduce the energy charge or the NADH/NAD+ ratio. When LEV or LND were given after ALA-PCT, these 2 treatment modalities acted in an additive or slightly synergistic manner.

CC 8-9 (Radiation Biochemistry)

ST energy metab inhibitor aminolevulinate photochemotherapy

IT Photosensitizers
(energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT Neoplasm inhibitors
(photosensitizing; energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT Phototherapy
(chemo-, energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT Animal metabolism
(energy, inhibitors; energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT Photodynamic action
(therapeutic, energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT 53-84-9, NAD 56-65-5, 5'-Atp, analysis 58-64-0, 5'-Adp, analysis 58-68-4, Nadh 61-19-8, 5'-Amp, analysis 86-01-1, Gtp
RL: ANT (Analyte); ANST (Analytical study)
(energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT 106-60-5, 5-Aminolevulinic acid 14769-73-4, Levamisole 50264-69-2, Lonidamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced photochemotherapy)

IT 553-12-8, Protoporphyrin IX
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FMU (Formation, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
(energy metabolism inhibitors lonidamine and levamisole effect on 5-aminolevulinic-acid-induced protoporphyrin photochemotherapy)

L63 ANSWER 22 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN

AN 1995:748075 HCAPLUS

DN 123:188214

TI Modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes

AU Comte, Blandine; Romanelli, Angela; Tchu, Sophie; van de Werve, Gerald

CS Dep. Nutrition, Univ. Montreal, Quebec, H3C 3J7, Can.

SO Biochemical Journal (1995), 310(1), 61-6

CODEN: BIJOAK; ISSN: 0264-6021
PB Portland Press
DT Journal
LA English
AB The mechanism of the antihyperglycemic action of dexfenfluramine (DEXF) was investigated in isolated rat hepatocytes exposed to glucagon. Preincubation of hepatocytes with DEXF caused a dose-dependent inhibition of cAMP formation by 100 nM glucagon (Ki = 0.29 mM) that was almost complete at 1 mM DEXF. Surprisingly, glucagon-induced phosphorylase activation was not affected by DEXF despite the significant drop in cAMP levels. Glucose production stimulated by glucagon was inhibited by ≤ 48% by 1 mM DEXF;, and the rate of glucose production correlated pos. with the steady-state concentration of glucose 6-phosphate. DEXF also partially restored lactate + pyruvate production which was abolished by an optimal concentration of glucagon. Although DEXF was not able to prevent the inactivation of pyruvate kinase by glucagon, the lack of further accumulation of phosphoenolpyruvate in DEXF-treated cells supports the conclusion that the flux through pyruvate kinase is stimulated, probably via the increase in fructose 2,6-bisphosphate, thereby increasing glycolysis. The results thus indicate that DEXF counteracts the inhibition of glycolysis by glucagon and that this property might contribute to the antihyperglycemic effect of this drug. Furthermore, this study shows that, in the presence of the drug, glucagon caused phosphorylase activation and pyruvate kinase inactivation without a significant increase in cAMP levels. Furthermore, this study shows that, in the presence of the drug, glucagon caused phosphorylase activation and pyruvate kinase inactivation without a significant increase in cAMP levels.
CC 1-10 (**Pharmacology**)
ST glucagon glucose formation dexfenfluramine hepatocyte
IT Antidiabetics and Hypoglycemics
Glycolysis
(**modulation** of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT Liver
(hepatocyte, modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT 9007-92-5, Glucagon, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT 3239-44-9, Dexfenfluramine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT **9001-59-6**, Pyruvate kinase 9032-10-4, Phosphorylase a
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT 50-21-5, Lactic acid, biological studies 50-99-7, D-Glucose, biological studies 56-73-5, Glucose 6-phosphate 60-92-4, CAMP 127-17-3, Pyruvic acid, biological studies 138-08-9, Phosphoenolpyruvic acid 79082-92-1, Fructose 2,6-bisphosphate

RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
IT **9001-59-6**, Pyruvate kinase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of glucagon-induced glucose production by dexfenfluramine in rat hepatocytes in relation to antihyperglycemic activity)
RN 9001-59-6 HCAPLUS
CN Kinase (phosphorylating), pyruvate (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L63 ANSWER 23 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1991:550834 HCAPLUS
DN 115:150834
TI Modulation of liver carcinogenesis by dehydroepiandrosterone
AU Mayer, D.; Weber, E.; Bannasch, P.
CS Inst. Exp. Pathol., Dtsch. Krebsforschungszent., Heidelberg, 6900, Germany
SO Biol. Role of Dehydroepiandrosterone (DHEA) (1990), 361-85. Editor(s): Kalimi, Mohammed Y.; Regelson, William. Publisher: de Gruyter, Berlin, Fed. Rep. Ger.
CODEN: 57DBAU
DT Conference
LA English
AB Administration of dehydroepiandrosterone (DHEA) to rats previously treated with chemical carcinogens modulates the process of hepatocarcinogenesis. The total number of tumors per animal is reduced compared to animals treated with the carcinogen alone, and these tumors exhibit a less malignant phenotype. The tumors are preceded by preneoplastic lesions of an amphophilic or amphophilic/tigroid appearance which do not or only very rarely occur in livers treated with the carcinogen alone. The amphophilic foci are characterized by alterations in the pattern of enzyme activities of carbohydrate metabolism which are less pronounced or even opposite to those observed in glycogen storage foci which usually represent the prestages of hepatocellular tumors in rat livers treated with the carcinogen alone. The **modulation** of metabolic pathways such as **glycolysis**, gluconeogenesis, or the hexose monophosphate shunt by DHEA may be related to the mechanism underlying the modulation of hepatocarcinogenesis by the hormone.
CC 2-4 (Mammalian Hormones)
Section cross-reference(s): **1**
ST liver carcinogenesis dehydroepiandrosterone
IT Liver, neoplasm
(dehydroepiandrosterone modulation of)
IT 53-43-0, Dehydroepiandrosterone
RL: PROC (Process)
(liver carcinogenesis modulation of)

L63 ANSWER 24 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1991:74777 HCAPLUS
DN 114:74777
TI Effects of the anti-AIDS drug dideoxyinosine on hepatic glycolysis in the perfused rat liver: role of intracellular calcium stores
AU Badr, Mostafa Z.
CS Div. Pharmacol., Univ. Missouri, Kansas City, MO, 64108, USA
SO Biochemical Pharmacology (1991), 41(1), 146-8
CODEN: BCPCA6; ISSN: 0006-2952

DT Journal
LA English
AB Therapeutic concns. of dideoxyinosine (ddI) stimulated glycolysis in the isolated, perfused rat liver. This stimulation occurred in the presence or absence of Ca2+ in the perfusate. In contrast, when intracellular Ca2+ stores were depleted, the drug caused only slight stimulation of hepatic glycolysis, which was restored to control values upon infusion of Ca2+ simultaneously with ddI. These findings suggest that ddI stimulates hepatic carbohydrate metabolism by mobilizing Ca2+ from intracellular stores. Stimulation of hepatic glycolysis, in conjunction with the poor nutritional state of AIDS patients, is expected to lead to the depletion of hepatic glycogen stores. Patients receiving ddI should be monitored closely for the earliest signs of hepatotoxic effects.
CC 1-5 (**Pharmacology**)
ST liver glycolysis dideoxyinosine calcium; carbohydrate metab liver dideoxyinosine calcium
IT Glycolysis
(by liver, dideoxyinosine effect on, calcium role in)
IT Liver, metabolism
(glycolysis by, dideoxyinosine effect on, calcium role in)
IT Carbohydrates and Sugars, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, by liver, dideoxyinosine effect on, calcium role in)
IT 7440-70-2, Calcium, biological studies
RL: BIOL (Biological study)
(**glycolysis** by liver response to dideoxyinosine **modulation** by)
IT 69655-05-6
RL: BIOL (Biological study)
(glycolysis by liver response to, calcium role in)

L63 ANSWER 25 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1981:150760 HCAPLUS
DN 94:150760
TI Monocyte-mediated antibody-dependent cytotoxicity. **Modulation** by **glycolysis** and insulin
AU Kragballe, Knud; Beck-Nielsen, Henning; Pedersen, Oluf; Ellegaard, Joergen; Soerensen, Niels Schwartz
CS Dep. Med., Univ. Aarhus, Aarhus, Den.
SO Scandinavian Journal of Haematology (1981), 26(2), 137-44
CODEN: SJHAAQ; ISSN: 0036-553X
DT Journal
LA English
AB In suspensions of purified human monocytes from 14 healthy persons, the antibody-dependent cell-mediated cytotoxicity (ADCC), the lactate [50-21-5] release, and the glucose [50-99-7] uptake were studied. In nonstimulated monocytes, ADCC correlated with lactate release and glucose uptake. Following addition of insulin [9004-10-8] a dose-related rise in ADCC, lactate release, and glucose uptake was observed For each of the 3 processes the maximal insulin effect was .apprx.30%. Most of the stimulation was seen within the physiol. concentration range of insulin, and the insulin concentration resulting in 50% of the maximal effect was nearly the same for ADCC, lactate release, and glucose uptake (.apprx.100 pM). The insulin stimulation of ADCC correlated with the stimulation of lactate release and glucose uptake. An inverse correlation between the ADCC of nonstimulated monocytes and the insulin stimulation of ADCC was demonstrated. No relation was found between monocyte maturity and any of

the 3 variables of monocytes function, either with or without insulin. Thus, for normal monocytes, the cytotoxic capacity is closely related to glycolysis.

CC 2-1 (Hormone **Pharmacology**)
Section cross-reference(s): 15

ST monocyte antibody cytotoxicity glycolysis insulin

IT Monocyte
(antibody-dependent cytotoxicity of, **glycolysis** and insulin **modulation** of)

IT Erythrocyte
(antibody-dependent toxicity to, monocyte in, insulin effect on)

IT **Glycolysis**
(by monocyte, antibody-dependent cytotoxicity **modulation** by)

IT 9004-10-8, biological studies
RL: BIOL (Biological study)
(antibody-dependent cytotoxicity and glycolysis response to, in monocyte)

IT 50-99-7, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, by monocyte, antibody-dependent cytotoxicity in relation to)

IT 50-21-5, biological studies
RL: BIOL (Biological study)
(release of, by monocyte, antibody-dependent cytotoxicity in relation to)

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